cost benefit of the programme could be quantified by a reduction in the need for hospital treatment, but there is little evidence to support this argument.

(8) What should be done about findings that are neither clearly normal nor obviously abnormal? Sickle cell trait clearly falls into this category. We now need to look further at how efficient our programme is since the introduction of citrate agarose electrophoresis to confirm that sickle cell trait is accurately and reliably detected. If so, we would then need to consider how this information could best be used. Counselling and follow up for all these babies would be a major undertaking and there are implications for the provision of prenatal diagnosis.

We use the neonatal capillary blood sample already collected for phenylketonuria and congenital hypothyroid screening. Thus the advantages are pre-existing blood collection, processing, and laboratory facilities and a negligible failure rate, unlike cord blood sampling.[5] The additional cost is kept to a minimum. Moreover, a computer system linked to the local child health register ensures that every baby is tested, although we have to record some babies as "not tested" because they have received a blood transfusion which would interfere with the interpretation. We are evaluating in the West Midlands Region which health districts could justify neonatal screening in this form. Several health districts have so few babies at risk that this is clearly not justified. In districts where there are few heterozygotes for sickle cell disease, identification and testing of the babies at risk only in each maternity unit will be more cost effective than comprehensive population screening.

Extending such a screening programme to other parts of the UK would depend upon the local incidence of sickle cell disease. In some regions an additional sample may be required as many screening laboratories use dried blood spot samples on filter paper cards for phenylketonuria and congenital hypothyroid screening. Although it is possible to elute haemoglobins from dried blood spots, the specimen is less stable and interpretation is more difficult.

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Medical History

Maurice Ravel's illness: a tragedy of lost creativity

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Abstract

Maurice Ravel had been subject to psychiatric disorder for many years when signs of organic brain disease appeared at the age of 52. Aphasia, apraxia, agraphia, and alexia became established some five years later. Musical creativity was lost. Alajouanine diagnosed cerebral atrophy with bilateral ventricular enlargement. Though Ravel's condition deteriorated progressively, generalised dementia was not apparent. He died in December 1937, after a craniotomy performed by Clovis Vincent, possibly from a subdural haematoma. Vincent's operative findings are described here. The likely cause of Ravel's illness was a restricted form of cerebral degeneration.

Observations on composers long dead are often of limited scientific value, for they depend on evidence from times when medical practice was less precise than it is today and when laboratory tests were virtually unknown. Thus although Meyer and Slater were able to construct an acceptable explanation of Robert Schumann's mental illnesses,[1] Henson and Urich found medical reports prepared in the early 1840s inadequate when they tried to assign a cause to the paralysis of the composer's hand.[2] Systematic examination of the nervous system had yet to become established. With composers dying in more recent years some difficulties of fact may be overcome, but other problems remain, including lack of important information—a natural deficiency in retrospective studies—and an understandable reticence of family and friends. Moreover, observations by untrained witnesses may be unreliable or misleading.

What is the justification for turning intimate personal matters into public property? We can fairly claim that knowledge of the sufferings of composers heightens our appreciation of them and their music. This carries no implication that neurological dissection of creative artists illuminates the sources behind the power of their art.

Maurice Ravel (1872-1937) developed progressive neurological symptoms over the later years of his life. The history of his consequent decline has been told by many friends and biographers. His recorded disabilities are susceptible to neurological analysis, though methods of studying the brain by imaging and biochemical techniques were largely undeveloped in the 1930s, and neuropsychology was barely poised for advance. There are conflicting and even erroneous published accounts of Ravel's illness, but it is now

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possible to reconstruct the history from newly available medical information and a recent comprehensive biography.[3]

All writers have agreed that Ravel's nature was private, enigmatic, and complex. There are indications of nervous disorder from before the first world war, but his experiences in that conflict (he was initially rejected for service on grounds of physique) and the death of his mother were aggravating influences from 1917 onwards. Important premorbid psychological factors included remarkable dependence on his family (especially his mother) and his compulsive or obsessional behaviour, evidenced by his self critical and fastidious nature from an early age, his compositional methods, and his collection of bibelots and mechanical toys. Ravel's output was not great, his perfectionism alone denied that, and he had his periods of compositional silence. Nevertheless he remained productive until some five years before his death in 1937.

Published studies

Information on Ravel's personality, behaviour, and medical history are contained in the biographies.[4-9] Marnat published a valuable comprehensive study,[3] bringing together information from diverse sources in a chronological order followed here. Among friends writing memoirs, Hélène Jourdain-Morhange[10] and Marguerite Long[11] proved perceptive witnesses, while Colette and others, "his companions in good and bad times," provided more general observations.[12] Gerar and Chalupt published a selection of Ravel's letters.[13] These are the main sources I consulted. On the medical side Alajouanine gave a conflation of his clinical observations,[14] while Vincent's operative findings are now known.

Medical history

Ravel was small in stature (1·60 m) with a large head. No measurements of his skull circumference have been found, nor are the results of radiographs of the skull known. Published serial photographs show few comparable views. The last studio portrait (1935) shows a handsome man with prominent frontal bones, not beyond normal limits, while earlier pictures show no obvious signs of hydrocephalus. Clovis Vincent thought that the size of the head was abnormal and apparently concluded that this was the result of hydrocephalus. With this interpretation the condition must have been compensated or arrested, given the earliest date—1927—for the onset of manifest neurological disorder when Ravel was 52. The nervous symptoms, mentioned above, and his recorded peculiarities of behaviour and temperament cannot be ascribed to organic brain disease. Ravel suffered various illnesses throughout middle age. He was seldom in good health from the first world war on, but he remained physically active. There is no indication that his neurological state was secondary to systemic disease. Dr Valéry-Radot, who cared for him over several years, wrote to Hélène Jourdain-Morhange in early 1934, "I have made numerous examinations to be sure there is no lesion whatsoever that has been overlooked. There is none there." Presumably he excluded the nervous system in writing thus.

The first clear signs of neurological upset appeared in 1927. Apparently Dr Valèry-Radot had already recommended a year's rest (the reasons for this advice have not been discovered), but Mme Jourdain-Morhange telephoned the doctor because she had found Ravel "so lost before his music—since 1927 at a concert where he was accompanying his violin sonata." Also he was making "blunders in writing, irregular lines and erasures (he who had always admired a clear hand)," and his hand trembled. Nevertheless his writing "was stabilised anew" in 1928. A highly successful American tour followed in the same year; Ravel had ignored his physician's advice. It appears that both Valéry-Radot and the patient already feared there was something serious amiss. In November 1928 Ravel lost his place when playing his *Sonatine* in Madrid—he jumped from the exposition of the first movement to the coda of the finale. Independent of this event his memory was said to be defective and his mental processes slowed. The course of the illness from 1929 to 1931 is not well documented, but during these years his last two major works, the piano concerti, were completed, while *Bolero* had received its first performance in November 1928, so invention had remained intact. It is notoriously difficult to ascertain the exact point of onset of progressive brain disease when the initial symptoms lie entirely within the mental sphere, but on the evidence, and despite the temporary restoration of the capacity to write, 1927 appears to be the relevant date. Mme Jourdain-Morhange sounded the alarm in that year, and she later wrote: ". . . at the beginning of his illness, Ravel flew into a passion when we could not find the word he sought," a sign of the aphasia which was developing. On 20 November 1931 he described himself as being "nearly finished" and said he was being treated with serum injections and complete rest. Yet he departed on a further prolonged tour with Mme Long immediately after the premiere of his piano concerto on 14 January 1932. Rubinstein described the strange episode on Prague station during the journey when Ravel abused Mme Long because she had mislaid their tickets.[15] Mme Long had herself noticed slowing of gestures and extreme lassitude.

Ravel was injured in a Paris taxi accident on 9 October 1932. Some teeth were broken, the head and chest were bruised, and he suffered shock. There is no evidence of any resultant neurological damage within the head, but it is common knowledge that ostensibly minor head injury can be linked with deterioration of brain disease already present (as was the case with Ravel).

In 1932 Ravel began work on the three songs *Don Quichotte à Dulcinée*. He was late in completion (which was not unusual), and the task was probably finished in early 1933. The orchestral holographs are in Ravel's hand, but Lucien Garban and Manuel Rosenthal assisted in transcription. The same friends wrote down the orchestration of *Ronsard à son Âme* to Ravel's "laborious dictation" in February 1933. This was the end of his creative activity.

Throughout 1933 matters grew worse. In June he tried to skim a pebble on the sea at St Jean de Luz but only succeeded in striking his childhood friend, Marie Gaudin, in the mouth. Previously a strong swimmer he lost the capacity altogether. Difficulty in writing increased, and he could not sign his name when making prints of his hands in November. According to Valentine Gross he said, "I cannot, I cannot sign. My brother will send you my signature tomorrow." His last published letter was written with great difficulty in February 1934.

Ravel was naturally alarmed by the illness which had robbed him of his capacity to compose and much else. Marnat described him as terrified because "he knew the fate of Baudelaire . . . equally haunting was the memory of his father's regression in 1908 and the fear of possible heredity." Mme Jourdain-Morhange wrote that he always hoped for a cure. Over the years he accepted a variety of unlikely remedies—"each friend proposed a sure means of cure. The desperate Ravel threw himself into the most unbelievable cures." In November 1933 he made his last public appearance conducting the piano concerto (with Mme Long as soloist) and *Bolero*. Marnat commented "without doubt the orchestra managed on its own." Piano practice had already ceased.

The remaining four years form a sad picture of progressive decline, although Ravel remained physically and socially active. His preserved (or largely preserved) auditory imagery meant that he could still hear music in his head. This must have encouraged his belief that he still had music to provide, as would the attested preservation of receptive aspects of musical function.

The neurological examination

Alajouanine's clinical findings were recorded in a Harveian lecture on aphasia and artistic realisation.[14] The subject and setting may explain the omission of normally expected information. Alajouanine followed Ravel's case for more than two years, but his observations were undated. It seems likely that they were covered by the years 1933-6; certainly they described advanced disability. The published translation from the French is less than satisfactory.

Alajouanine wrote "at the peak of his artistic achievement . . .

Maurice Ravel is struck down by an aphasia," a statement which does not necessarily imply an acute onset. The findings are summarised here. There was a marked ideomotor apraxia, and this affected Ravel's capacity to write, both in verbal and musical terms, and to play the piano. (There is a document in existence, dated February 1936, showing gross impairment of Ravel's capacity to write both his own name and musical notation.) There was moderate impairment of the production, and less so the comprehension, of the spoken word. Reading and writing musical notation were "very difficult" and copying almost impossible. His capacity to play the piano was virtually lost. By contrast, Ravel's auditory perception was relatively preserved. He noticed that Alajouanine's piano was out of tune, and his capacity to reproduce notes played on the piano was "quite good." He recognised most works previously known when they were played to him (surely he would have recognised them all if his musical memory had been intact), and he was faultless in identifying his own compositions. He was quick to notice deliberately introduced mistakes in *Le Tombeau*, and also identified faulty rhythms and tempi. Alajouanine thought that Ravel's memory, judgment, and affective and aesthetic responses were intact, though the patient must have had difficulty in expressing both judgments and responses verbally.

Alajouanine concluded that the cause of Ravel's condition was cerebral atrophy, the condition was "quite different from Pick's Disease," though he did not say why. He also said there was bilateral ventricular enlargement, an observation which could only have been surely made on radiological grounds, so air studies were probably performed.

Ravel suffered aphasia, apraxia, agraphia, and alexia. On the musical side he could not read effectively, write notation, use musical signs, nor play the piano. Tonal recognition and musical memory and imagery were relatively preserved. Peculiarly, Alajouanine did not use the long established term amusia in his report. This ugly word is used to describe impairment or loss of musical function deriving from acquired disease of the brain. Amusia occurs characteristically with damage to certain localised parts of the brain.[16,17] The combination of Ravel's defects shows that the dominant perisylvian region of the brain was affected. Alajouanine did not mention Ravel's handedness, a strange omission, but the evidence suggests that he was right handed and that the constellation of symptoms was due to damage to the left side of the brain.

Alajouanine regarded Ravel's creative failure as an inability to realise or communicate his musical thoughts by notation or play and this must be true, although it was an oversimplification. Ravel indicated that although he could hear music in his head he could not communicate these experiences to others. There lay the tragedy: in Alajouanine's words, "to conceive is nothing, to express is all." Naturally, there are no means of assessing the quality of Ravel's musical thought or potential creativity at this time. With current methods of neuropsychological examination more might have been learned, but in the light of Alajouanine's findings, and the defect of musical memory noted above, it seems unlikely that the quality of his musical thought approached his normal standard at this time.

Roughly three quarters of all amusic patients are also aphasic, but conversely the vast majority of aphasic patients are not amusic. On this and other grounds we conclude that although speech and music are closely linked they have different substrates in the brain.[18] Maurice Ravel is the only important composer known to have suffered the indignity of amusia. Shebalin had a similar speech disturbance from a stroke, but he was not amusic and was able to continue composing.[19] There are superficial resemblances between Ravel's case and that of Benjamin Britten, who suffered a stroke about the time of a heart valve replacement (M C Petch, personal communication, 1987). Britten's subsequent difficulty in notating a score sprang initially from weakness and loss of sensation in the right upper arm. The only persistent handicap was a stiffness of the right shoulder (a complication of the stroke), which made it difficult for him to write a full score (personal communications: P Pears, 1985; I Tait, 1987; D Thompson, 1987). He suffered briefly from aphasia, but there is nothing to suggest amusia. Indeed, his restricted output in his last years was determined by the severe heart failure from which he died (personal communications: M C Petch, 1987; I Tait, 1987).

Marnat painted the sad picture of Ravel's further decline from the histories recounted by friends and summarised the available medical information. Colette remembered how he arrived on foot at the Moreaus' house, near Montfort, after dinner in the summer of 1937. On seeing her he said, "Tiens, Colette," but otherwise "scarcely attempted to speak . . . . he appeared as if about to break up." The last concert at which he assisted (as répétiteur) took place in November. At this time his old friend Ida Rubinstein, the dancer, dismayed at the relentless progression, consulted Thierry de Martel, the distinguished French surgeon, and he disapproved of the idea of neurosurgical intervention. She then sought further opinions in Switzerland, Germany, and England, but "everyone thought there was no tumour, but a degenerative condition."[3] Clovis Vincent, the well known Paris neurosurgeon, was then approached, and he advised surgical treatment "in case there was a tumour." Furthermore, he thought that the ventricular dilatation (which he believed or knew to be present) was not "due to a true atrophy, that it had increased with age, and that an operation might prevent progression." Edouard Ravel, brother of the composer, accepted this advice; the patient was clearly in no condition to express a considered view.[3] Mme Long wrote that Ravel's friends were against surgery.

There are no notes available to show just what was in Vincent's mind. On Wednesday, 17 December, according to the notes, he operated on a diagnosis of ventricular dilatation, presumably cause undetermined, though it seems that Ravel's large head formed one basis for his opinion. The skull was opened by a *right* frontal bone flap, presumably to avoid surgical damage to the dominant hemisphere. No bony abnormality was remarked. The brain was found to be slack, without softening in the area displayed. Puncture of the right lateral ventricle produced no spontaneous flow of cerebrospinal fluid but fluid escaped with pressure on the brain surface. Vincent injected water into the ventricle in an attempt to raise the pressure, but the manoeuvre was unsuccessful and he prudently decided to withdraw. The skull flap was replaced, leaving the dura open, and the wound closed. He had found no abnormality beyond the subatmospheric intraventricular pressure. He did not proceed to ventriculography, which was wise, nor did he make a biopsy; there was no visibly abnormal brain.

After the operation Ravel's friends were cheered by an apparent improvement in his condition, but this was short lived as he soon lapsed into coma and died on 28 December. Permission for a necropsy was not obtained so the immediate cause of death was not determined. Retrospectively, it is likely that surgical interference in a case of low pressure ventricular enlargement led to formation of a subdural haematoma and unrelieved cerebral compression.

Diagnosis

What was the nature of the original brain disease that caused Ravel's decline? The absence of a necropsy means the diagnosis will never be certainly known. The slow evolution of the condition suggests a progressive degenerative complaint. Alajouanine is said to have thought initially that Ravel suffered a form of stroke, but he later concluded that the complaint was a cerebral atrophy or degeneration, and this must be the favoured answer. Both Valéry-Radot and Alajouanine had excluded systemic disease, either specifically or by inference. Unfortunately, no records of blood pressure, urine analysis, and other laboratory tests have been found.

The commonest cause of progressive cerebral degeneration in late middle age is Alzheimer's disease, which is occasionally hereditary. Despite Ravel's fears, evidence that his father suffered from the complaint is at present unconvincing. In a few cases the disease presents with language disorder, which may remain the sole or dominant feature for some years. Given the duration of Ravel's illness, and the continuance of entirely focal symptoms for so long, Alzheimer's disease becomes a less attractive diagnosis, though it cannot be ruled out. Pick's disease is an alternative answer, though Alajouanine's exclusion (possibly on pneumoencephalographic

grounds) must carry weight. In fact Alzheimer's and Pick's diseases may be differentiated only by histological examination of the brain.[20 21] Mesulam described six patients who suffered slowly progressive aphasia, without generalised dementia[22]; he thought they might constitute a syndrome of relatively focal cerebral degeneration, with a predilection for the left perisylvian region. Others have described similar cases.[23] On clinical grounds Ravel's case falls into this group, the pathology of which has now been described in two patients who came to necropsy.[24] Both showed a focal, spongiform cortical degeneration involving the left inferior frontal gyrus, while in one the left superior temporal gyrus was also affected. In neither case were changes characteristic of Alzheimer's, Pick's, or Creuzfeldt-Jakob diseases found. Mesulam discussed the differentiation of primary progressive aphasia from Alzheimer's disease.[25] Vascular disease can be excluded, while the evidence is clearly against tumour. The ventricular enlargement diagnosed by Alajouanine and Vincent can be ascribed to atrophy. Under these circumstances the intraventricular pressure would have been normal or low.

Conclusion

As to the medical men engaged in this last Affaire Ravel, Alajouanine was already established as a neurologist, later to become the doyen of French neurology, while Vincent was a French neurosurgical pioneer. There was nothing either could have done to arrest or cure Ravel's condition. With modern methods of study to help him Vincent would not have operated. The task of managing a distinguished patient with irremediable progressive brain disease is never easy, and pressure for contraindicated intervention can be strong. Ravel's case illustrates this. Vincent failed to obtain the necropsy which would have established the diagnosis, and so the exact nature of the disease which destroyed this talented, ironic, enigmatic private man can only be inferred—and perhaps this is right.

After Ravel's death the person appointed guardian of the museum, which his home at Montfort l'Amaury became, was Celeste Albaret, Proust's housekeeper for many years. This choice made an appropriate ending to a sad tale of lost creativity. No wonder Ravel's family and friends were disturbed and horrified as they witnessed its unfolding. And what of ourselves? Exploration of this history indeed heightens appreciation of Ravel's artistry when his musical mind was intact and leaves us to reflect on the nature and extent of the loss to music which his premature disablement and death entailed.

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MATERIA NON MEDICA

Nothing if not critical

When we submit, publish, or deliver papers we get reports from referees and comments from letter writers or from the floor—lauding, approving, denegrating, or demolishing. We may also express ourselves on others' work in diaries or in personal correspondence, with varying degrees of detachment or of vehemence. How have musicians, artists, and poets reacted to the works of their colleagues?

In his diary Tchaikovsky commented, "I like to play Bach because it is interesting to play a good fugue; but I do not regard him as a great genius." He wrote, moreover, "I played over the music of that scoundrel Brahms. What a giftless bastard." Also on Brahms, George Bernard Shaw wrote in a letter, "Brahms is just like Tennyson, an extraordinary musician with the brains of a third-rate village policeman." Sir Thomas Beecham, the great conductor, wrote of Bruckner, "In the first movement alone of the Seventh Symphony, I took note of six pregnancies and at least four miscarriages." A critic in the *Dramatic and Musical Review* held that "Berlioz, musically speaking, is a lunatic; a classical composer only in Paris, the great city of quacks. His music is simply and undisguisedly nonsense." More forcefully written was a review in the *Chicago Tribune*: "The music of *The Love of Three Oranges*, I fear, is too much for this generation . . . Mr Prokofiev might well have loaded up a shotgun with several thousand notes of various lengths and discharged them against the side of a blank wall."

Turning to artists, to William Blake's eye "Rubens's colouring is most contemptible. His shadows are filthy brown, somewhat of the colour of excrement." Whistler said, "Rossetti is not a painter. Rossetti is a lady's maid." Of Whistler's *Nocturne in Black and Gold* Ruskin wrote, "I have seen and heard much of cockney impudence before now, but never expected to hear a coxcomb ask two hundred guineas for flinging a pot of paint in the public's face." Igor Stravinsky, on being detained by Italian border guards in 1917, reported, "I was accused of trying to smuggle a plan of fortifications—in fact my portrait by Picasso—out of Germany."

Of poets and writers, Virginia Woolf wrote, "Fate has not beeen kind to Mrs Browning. Nobody reads her, nobody discusses her, nobody troubles to put her in her place." In turn, Edith Sitwell wrote of Virginia Woolf, "I enjoyed talking to her, but thought *nothing* of her writing. I considered her a beautiful little knitter." And of Edith Sitwell, Dylan Thomas wrote in a letter to Glyn Jones, "So you've been reviewing Edith Sitwell's latest piece of virgin dung, have you?" Samuel Johnson remarked, "*Paradise Lost* is a book that, once put down, is very hard to pick up again." Of Johnson himself, Thomas Jefferson wrote, "Take from him his sophisms, futilities and incomprehensibilities, and what remains? His foggy mind." In his forthright manner Lord Byron wrote, "Here is Johnny Keats's piss-a-bed poetry. No more Keats. I entreat: flay him alive." Shelley, in his preface to *Adonais*, wrote of Keats, "The savage criticism of his *Endymion* . . . produced the most violent agitation in his susceptible mind . . . ended in the rupture of a blood vessel in the lungs; a rapid consumption ensued with early death at 28 years." We, as researchers, can be, and on occasion are, savaged by critics, but not to the extent of poor Keats.—ALEXANDER R P WALKER, Johannesburg, South Africa.

[All quotations are from *Pepper's Dictionary of Biographical Quotations*. (London: Sphere Books, 1985.)]